

Nuclear Radiation in Warfare

Dr ofstedal

FEB 22 1982

R.S.

This appraisal on genetic effects, by Dr. Rotblat, has just come to my attention. It paints a picture very similar to my own conclusions. The uncertainties of estimate could of course be in either direction, but for human risk are bounded by the empirical studies in Japan.

I hope this is useful

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Taylor & Francis Ltd
London



Oelgeschlager, Gunn & Hain, Inc.
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3.4.4. Effects of pre-natal exposure

The human embryo and foetus are highly sensitive to radiation and a number of deleterious effects were observed in children born to mothers who were exposed to ionizing radiation during pregnancy, at doses much lower than might have been expected from observations on adults.

Among the women who were pregnant during the explosions in Hiroshima and Nagasaki there was a marked increase in still-births, and the mortality of their children during the first year of life was considerably increased. The surviving children showed a greater frequency of mental retardation and they had head circumferences which were smaller than normal; other malformations included defects of the skeleton and the eyes. The stage of gestation at the time of exposure influences the type and frequency of the impairment. The risk of malformation is largest in the early stages of pregnancy but the risk of pre-natal death increases in the later stages.

Exposure in pregnancy also increases the rate of induction of cancer in children born to these mothers. Even a diagnostic dose of about 10–20 milligrays may increase the incidence of leukaemia and other cancers by some 50 per cent. These cases of leukaemia occur between three and eight years of age, with a peak at about five years. It is not yet known whether the incidence of leukaemia will rise again in adult life. The absence of an increase in cancer rate among children born to women in Hiroshima and Nagasaki who were pregnant during the explosions is one of the odd features of these survivors.

3.5. Genetic effects

If the germ cells of a person—as distinct from the somatic cells—receive a dose of ionizing radiation, changes may occur which would manifest themselves in the offspring of the exposed person or in future generations; these are referred to as genetic effects. Two types of damage may occur, gene mutations—that is, an alteration in the structure of one of the genes—and chromosome aberrations, which may affect a number of genes at the same time. It is generally accepted that both types of change are harmful to the descendants, but the type and magnitude of the effects vary enormously, from barely detectable to lethal in their consequence. In the latter case, if the result of the genetic change is death *in utero* or in early life, this particular type of defect will be removed from the genetic pool of the population. But non-lethal defects will be carried from generation to generation, particularly when the welfare society enables affected individuals to survive to maturity and beget children. Some of the changes, known as recessive mutations, reveal themselves only when both parents happen to have had the same mutation. These mutations may thus be

carried through a number of generations before the effect of the irradiation becomes manifest.

Any genetic defect that may result from exposure to radiation is likely to be the same as one produced by other mutagenic agents and which already exists in the population. About 10 per cent of all live births carry a significant genetic defect and exposure to radiation may be expected to add to this burden an amount dependent on the dose. The genetic effect of radiation is often expressed in terms of the 'doubling dose', that is, the dose of radiation which would add to the genetic pool the same number of defects as occur naturally. The concept of a constant doubling dose implies linearity between effect and dose. However, the presentation of the genetic effect by a single linear relationship can only be an approximation. Experiments with mice have shown that different genes often differ in their sensitivity to radiation. There is also a dependence on dose rate which is different for males and females. The doubling dose would thus be a value averaged over many conditions.

Even with this qualification there is a large uncertainty in the value of the doubling dose. For man it is taken to be between 0.5 and 2.5 Gy, but it may well lie outside these limits. The reason for this large margin of error is that the doubling dose has to be extrapolated from data on animals, mainly mice, as there are no human data available.

The only irradiated population which was thought to be large enough to show genetic effects are the survivors of the A-bombs in Japan. But despite a prolonged investigation of the first generation of children born to the survivors no increase in the incidence of genetic damage has been established. The effects studied included the incidence of abortions, still-births, congenital defects, infant mortality, sex ratio of children and birth weight, as well as direct measurements of the incidence of chromosome aberrations. In none of these criteria was the difference between the exposed and control populations statistically significant.

Different interpretations of this negative finding were put forward: (a) that the methodology used in the investigations was faulty; (b) that the sensitivity of the criteria used was too low for the size of the population investigated; (c) that the survivors are not a typical population as far as radiation effects are concerned; (d) that a genetic effect does exist but with a lower frequency than deduced from animal experiments; and (e) that there is no genetic effect of radiation in man.

This last interpretation cannot be taken seriously, but it is being used in popular publications to convince the public that there is no reason to worry about genetic effects of radiation. Genetic effects of radiation have been observed in animal species and there is no reason why the human species should be unique in this respect. Nevertheless, the absence of any effect is puzzling. The ICRP has used a risk factor for genetic damage in the first two generations of 4×10^{-3} per sievert; this was based partly on animal data and partly on the lower limit of the doubling dose that can be deduced from the negative findings in Hiroshima and Nagasaki. But like the somatic risk factors, the ICRP figure for genetic damage may be wrong by a large factor.